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Effect of very high rearing temperature on changes in hormones responsible physiological stress in broilers reared in western Algerian

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Article Info	Abstract
Keywords : Algeria Climate Hormonology Poultry Resistance Scorching heat *Correspondance : attousahnoun@hotmail.fr Reçu le 31 mai 2018, Révisé le 17 juin 2018, Accepté le 18 juin 2018.	This study was conducted to evaluate the effects of a very hot temperature on broilers. Faced with this severe heat, we wanted to determine the limits variations in growth performance in broilers. Above all, it was of interest to determine whether or not the hormones triiodothyronine (T3), thyroxine (T4), aldosterone (Ald) and ACTH would also be affected by changes resulting from very hot. Two hundred male chicks (Hubbard) of a day were reared during 50 days. They were divided into two groups. Two thermal environments were applied from the 7th day of rearing: Thermoneutral (NT: 25 °C) for the first group and a very hot temperature (HT: 37°C) for the second. Every day, broiler chickens received ad libitum feeds and water drink. DRG® ELISA (Enzyme linked Immuno Sorbent Assay) kits were used for the determination of T3, T4, Ald and ACTH (EIA-3801, 4568, 4128 and 3647 respectively). Decrease in the feed intake (FI) due to HT was observed. Superiority of body weight (BW) in NT broilers was recorded. A significantly higher deposit of subcutaneous lipids (SCL) up to 105g/Animal was obtained in HT broilers. Weight of abdominal fat (AF) was higher in NT broilers. From day 18, T3 decreases were recorded in HT broilers. At the end of the breeding, the level of T3 in HT broilers was identical to that of NT broilers. The HT broilers had recorded higher levels of Ald. From the 40th day, significant increases in ACTH levels were recorded in HT broilers.

1. Introduction

In western Algerian, poultry faces several problems such as climatic conditions. Indeed, summer temperatures often very high and persistent are a real obstacle to the success of poultry farming. Because of mortality, these temperatures can cause significant economic losses. Economic losses caused by heat relate laying hens [21] and broilers [10]. Because of this problem, several studies on the heat acclimatization of the chicks were performed. Generally, these studies were conducted over short or medium exposure duration to heat [1, 38, 6, 33].

Further studies were performed to determine the effects of heat on the production parameters and poultry physiology [2, 12]. In addition, the temperatures tested by these authors were moderately hot compared to those recorded in Algeria, especially in summer.

Because of these differences between the temperatures already tested, we tested other more severe thermal conditions and similar to the Algerian reality [4]. In these thermal conditions, we recorded large changes in performance in broilers [4]. However, some questions remained unanswered to better explain the changes induced by the high heat. So, we wanted to determine how to vary the main hormones responsible for the physiological stress (Ald and ACTH) [22, 23] and growth (T3 and T4) [17].

For this study, we extended the same experimental conditions as those applied by Attou *et al.* [4]. In the thermal conditions in which broilers were reared were severe. They are similar to environmental conditions poultry farms in Algeria. From a young age until the end of breeding, the animals were raised in a temperature of 37 °C.

The main objective of this study was to determine how the animal reacts to this extreme

heat throughout the rearing period: Especially if certain hormones undergo changes? The hormones involved in this study are T3, T4, aldosterone and ACTH considered primarily responsible for stress and growth.

2. Materials and methods

2.1. Animals and diets

Two hundred male broilers chickens Hubbard aged one day were raised during 50 days. The chicks were divided into 2 groups, each group contained 100 animals.

Just after hatching, the chicks were vaccinated against Marek disease and infectious bronchitis. After one, two and three weeks of age, they had received in the drinking water vaccination against Newcastle disease associated with a triple vaccine (respiratory disease, Gumboro and infectious bronchitis).

Two ambient temperatures were applied from the 7th day of rearing. The first group was exposed to thermo neutral temperature (NT: 25°C) and the second group at a very hot temperature (HT: 37°C). The average weight of chick was 39.2±3g. The diets characteristics were presented in **Table. 1**. Every day, the animals received ad libitum feeds and water drink.

The measures of feed intake (FI), body weight (BW), eviscerated carcass (EC), abdominal fat (AF) and subcutaneous lipids (SCL) were determined on the 50th day.

At 18, 25, 32, 40 and 50 days of age, other broilers were bled and blood samples were made for analyzes of T3, T4, aldosterone (Ald) and ACTH.

Table 1: Compositions and nutritional characteristics of diets.

Ingredients (g/kg)	Starter diet	Grower diet
Crude Protein	200	190
Fat	35.6	34.9
Crude Fiber	27.7	28
Ashes	43	41.6
Moisture	55	60
MVC ¹	10	10
L-Méthionine	0.3	-

¹Mixture Mineral and Vitamin (mg/kg diet): Fer: 60; Brass: 10; Zinc: 80; Manganese: 80; Cobalt: 0.2; Selenium: 0.2; Iodine: 1; Vitamin E: 15; Menadione (K₃): 5; Thiamin (B₁): 3; Riboflavin (B₂): 7; Pantothenic acid (B₅): 10; Niacin: 30; Folic Acid (B₉): 0.5; Vitamin B₁₂: 0.02; Pyridoxin (B₆): 4; Choline chloride: 300

2.2. Laboratory analysis

The crude protein of diets was determinate by the Kjeldahl method [3]. The concentration of fiber diets was determinate with the Weende method after a double hydrolysis of non-cellulosic constituents in acid solution and in the other basic respectively [3]. The content of lipids diets was determined by extraction in a dispositive SOXHLET

by the petrol ether [3]. The subcutaneous lipids were determined after extraction with chloroform-methanol (2v/1v) [11].

DRG® (Enzyme linked Immuno Sorbent Assay) kits were used for the determination of hormones. For analysis of T3 and T4, we used a solid phase competitive enzyme immunoassay (EIA-3801 and EIA-4568 respectively). The antibodies used were Rabbit anti-Sheep IgG antibody and donkey anti-mouse IgG antibody (Respectively for T3 and T4). The analysis of Ald follows the principle of ELISA according to the model of competitive binding (EIA-4128). Rabbit anti-aldosterone antibody was used. The ACTH immunoassay test was a two-site ELISA for the measurement of the biologically active 39 amino acid chain of ACTH (EIA-3647). A goat anti-ACTH antibody and a mouse anti-ACTH antibody were used.

2.3. Statistical analysis

The statistic was undertaken by analysis of variance (ANOVA) in two factors of variation (temperature and age) with XLSTAT. The Newman-Keuls test was applied for comparison of mean values. Each mean value is assigned a letter; means followed by different letters are significantly different. Difference between two means was considered statistically significant at $p < 0.05$. Microsoft Office Excel (2007) was used for the histograms of figures.

3. Results

Some results of growth performance and carcass parameters are shown in **Table. 2**. A decrease of 18% of the FI was recorded in HT broilers. A significant decrease ($p < 0.05$) of BW in HT broilers was observed at the end of breeding. Otherwise, the HT had a significant negative effect ($p < 0.05$) on the EC with a decline of 15%. This allowed EC/BW lower in HT broilers. The HT caused a significantly higher SCL at the end of rearing. What caused the EC oilier with SCL/EC significantly higher. Conversely, the AF was significantly greater in NT broilers.

Table 2: Feed intake (FI), body weights (BW), eviscerated carcass (EC), subcutaneous lipids (SCL) and abdominal fat (AF) from broilers reared at the very hot temperature (HT) and thermoneutral temperature (NT).

Parameters	Age (day)	Rearing Temperatures (°C)	
		NT	HT
FI (g/d) (n=30)	50	141	119
BW (g) (n=30)	50	2142 ^a ± 108	1937 ^b ± 139
EC(g) (n=10)	50	1620 ^a ± 65	1373 ^b ± 95
EC/BW (%) (n=10)	50	75.28 ^a ± 3	71.47 ^b ± 6
SCL/EC (%) (n=10)	50	4.1 4 ^b ± 0.2	7.1 ± 0.6 ^a ± 0.3
AF (g) (n=10)	50	38.31 ^a ± 2.50	31.73 ^b ± 3.23

Results expressed as mean and standard deviation (SD).
a, b: values with different letters differ significantly ($p < 0.05$).

From 18 days, significant decreases in T3 were recorded in HT broilers (**Fig. 1**). These decreases vary according to the age of broilers. These decreases had reached high values of approximately 50, 25 and 71% respectively in the 25th, 32nd and 40th days. At the end of the rearing T3 in NT broilers had been a significant increase and become equivalent to that of HT broilers.

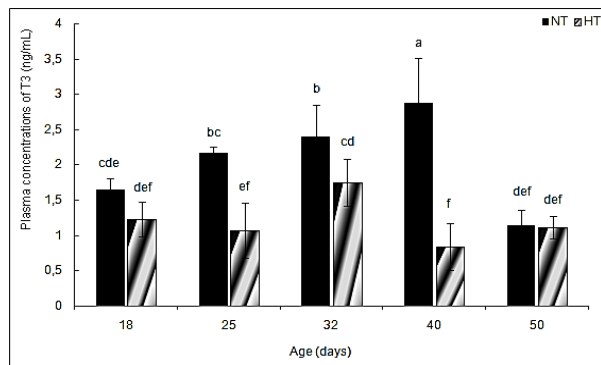


Fig. 1: Plasma concentrations of triiodothyronine (T3) in Broilers reared at the very hot temperature (HT) and thermoneutral temperature (NT). Data are means of 7 replicates (n=7).

Such as T3, T4 were significantly decreased ($p < 0.05$) from the 25th day in HT broilers (**Fig. 2**). Compared to NT, the decrease in T4 has reached 39% in the growth phase of HT broilers (25th day). She was only 16% in the finishing phase (50th day).

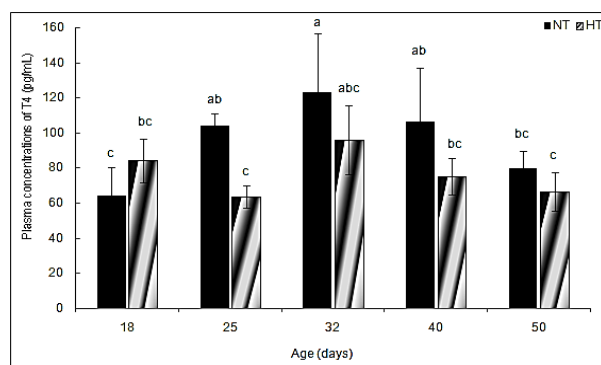


Fig. 2: Plasma concentrations of thyroxine (T4) in Broilers reared at the very hot temperature (HT) and thermoneutral temperature (NT). Data are means of 7 replicates (n=7).

The broilers HT showed levels of Ald significantly ($p < 0.05$) higher than that of NT broilers, except at 32 days of age (**Fig. 3**). At the beginning of the growth phase, these increases were approximately 30% in the 18th and 25th day. At 40 and 50 days, moderate increases of Ald caused by HT were recorded. Whatever the temperature applied progressive and significant ($p < 0.05$) increases of Ald

levels were recorded until day 32. Then they decreased significantly for both temperatures.

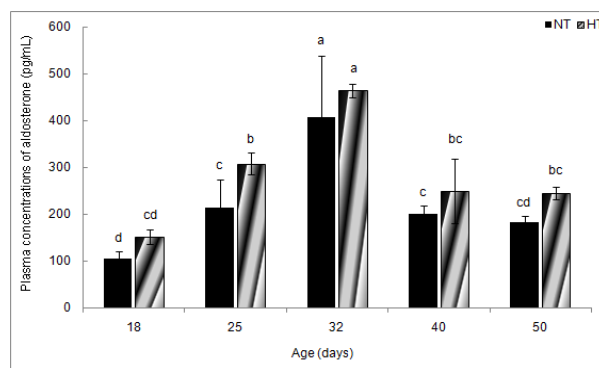


Fig. 3: Plasma concentrations of aldosterone (Ald) in Broilers reared at the very hot temperature (HT) and thermoneutral temperature (NT). Data are means of 7 replicates (n=7).

Until the 32th day, no temperature effect on ACTH was observed in broilers (**Fig. 4**). From the 40th day, significant increases ($p < 0.05$) of ACTH caused by HT were recorded. These increases were 92 and 39% in broilers 40 and 50 days of age respectively. However, for both rearing temperatures no significant increases of ACTH were noted with age until day 32. However, the increases become significant from 40th day in HT broilers.

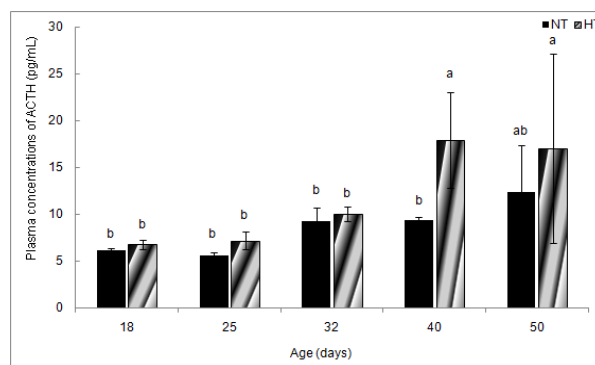


Fig. 4: Plasma concentrations of ACTH in Broilers reared at the very hot temperature (HT) and thermoneutral temperature (NT). Data are means of 7 replicates (n=7).

4. Discussion and conclusion

The very hot temperature caused a depressive effect on T3 by decreasing its concentration in broilers during the two phases (growing and finishing). Our results agree with those of other authors [36, 20]. During the growth of broilers, heat stress of short duration (35 °C or 36.5 °C; 6-24 h) may cause a decrease in T3 [36, 20]. By cons, a less intense heat (28 or 30 °C) has a limited effect on T3 [37].

The thermal effect on thyroid hormones was more noticeable at higher heat. Indeed, significant decreases in T3 were already reported by Kataria *et*

al. [17] exposing broilers at temperatures of 42-45 °C. According to Sokolowicz & Herbut [27], the decrease of T3 could be related to low metabolic activity followed by better thermoregulation. This physiological state condition protects birds against hyperthermia.

The decrease of FI observed in HT broilers corroborates the results of Attou *et al.* [4]. It could be attributed to the decrease of T3. Previously, this observation was reported by Carew *et al.* [5] who found a correlation between decrease FI in the heat and thyroid hormones. The same correlations between FI and T3 were obtained by Yahav [37] in broiler chickens and turkeys placed in temperatures 28 to 30 °C.

In addition, the decrease thyroid hormone was followed by a decrease in BW of HT broilers. Even depressive effect of HT on BW was found by Attou *et al.* [4]. This could justify the role of thyroid hormones in the growth process of animals. Our results corroborate those of Yahav *et al.* [35] who were unanimous on the direct or indirect involvement of heat on the altered of T3 and consequently decrease the growth of broilers. However, these changes differ from one environmental condition to another, i.e. an experimental model to another. This decline in growth caused by heat and thyroid hormones may be the result of a temporary or chronic disruption of metabolic activities, depending on the intensity and duration of heat stress.

Moreover, the involvement of T3 in the growth process was highlighted by the thyroid status of the animals by the hormonal treatment. Thus, an association between growth and T3 was observed earlier in the thyroidectomized chicks [13]. Higher levels of T3 in the fast-growing broilers were reported by Lauterio *et al.* [18], which suggests the important role of the T3 in the synthesis metabolism. Moreover, it has been established that the administration of T3 in animal increases its oxygen consumption and heat production [34]. By cons, hypothyroidism causes opposite effects [14].

Changes of thyroid hormones caused by HT could have indirect effects on intestinal function. Remember the existence of correlation between FI and T3 mentioned above. This suggests morphological changes in the small intestine and new dynamics of enterocytes that result in changes in FI then influencing T3. After administration in animal, T3 could lead to increased cell proliferation and the activity of Brush Border Membrane and the thickness of intestinal mucosa [15, 16]. I.e, it is likely that the HT and FI reduction caused a decrease in T3. In turn, decrease of T3 could alter the proliferative capacity of the enterocytes. Accordingly, this results in a decrease of intestinal absorption. Decreases in T3 content in HT broilers might be related with the decrease of AF and increase of SCL.

The relationship between thyroid gland and adiposity has been already reported by Decuyper *et*

al. [7] who observed a decrease in AF of broilers treated with T3 or T4.

Our results also corroborate those of Stewart and Washburn [29] who found a negative correlation between T3 and carcass fat of broilers. Finally, whatever direct or indirect involvement of T3, high heat is the trigger of this process of adiposity, with a limited AF and increased SCL.

Simultaneous declines of T3 and T4 caused by the HT assume that there is a relationship between these two hormones. This relationship is explained by the renewal of the T3 when the body needs this hormone. As in mammals, three deiodinase enzymes are responsible for the conversion of T4 to T3 in birds [8]. This is an Outer-Ring Deiodinase-1 (ORD-1) that converts T4 to T3 biologically active. Second, the ORD-2 which also converts T4 to T3 and whose action is mainly in the brain (also present in other peripheral tissues such as liver). Third, the Inner-Ring Deiodinase-3 (IRD-3) which degrades T3 to T2 or converts T4 to reverse-T3. Thus, 3,3',5-Triiodothyronine (T3 active), considered the main cause of biological actions, comes mainly from the peripheral deiodination of T4 by ORD-1 in the liver, kidney and intestines [8, 24].

It is likely that during chronic exposure of chickens to HT, the T3 had continued to decline. But at the same time, the conversion of T4 to T3 by the ORD-1 remained insufficient. What had led to lower T3 levels in HT broilers. However, the balance between T3 and T4 could not be obtained until the end of rearing. This discrepancy between the decrease in T3 and T4 conversion has already been observed in broilers exposed to heat [25, 28, 30]. We suggest that discrepancies between T3 and T4 also result from the difference between the experimental models. Indeed, after exposure of broilers to a cyclic heat (heat cycles), T4 response to his conversion was relatively slow and did not begin until the second day of heat exposure [30].

Changes in Ald caused by heat coincide with two important phases of rearing (growing and finishing) during which the body is subjected to an intense metabolic activity. The intervention of an environmental factor such as high heat could cause a hormonal upheaval allowing the animal to adapt and/or resist this new physiological situation. Add to Ald another hormone could suffer the same changes. This hormone is ACTH. Indeed, the same trends to increased ACTH were observed in HT broilers especially at the end of growth and during finishing. Our results agree with those of Deyhim & Teeter [9] who noted an increase in Ald in broilers exposed to cyclic summer temperatures 24-35-24 °C. They also agree with those of Kataria *et al.* [17] who observed a strong increase in Ald in broilers after exposure to elevated temperatures (42-45 °C)..

A simultaneous increase of Ald and ACTH is serious evidence of physiological stress in chickens, especially in old age [22, 23]. These two hormones intervene in perfect synergy. Their increase caused

by HT was presumably followed by a succession of physiological changes. These physiological changes had allowed the animal to adjust its metabolism to resist and/or adapt to this environmental factor that high heat. Among these changes, some aspects have already been mentioned by Attou *et al.* [4]. Moreover, a decrease of growth in broiler after administration of ACTH or exposure to heat was reported by Thaxton *et al.* [32], Puvadolpirod & Thaxton [22] and Lin *et al.* [19]. Also, a disturbance followed by a decrease in FI was observed by Zachariasen [39] in chicks treated with ACTH. This decrease of FI was also followed by increased consumption of water (polydipsia) and polyuria during physiological stress. These results could probably explain the decrease in the FI of HT broilers and their behavior to cluster around the waterers during the growing and finishing phases.

According Tankson *et al.* [31], broilers placed in a hot environment (24-34 °C) and treated with ACTH showed a decrease in their BW and muscle protein content of carcass. Remember that Attou *et al.* [4] had also found a decrease in BW broilers reared in 37 °C. This justifies that combined effect of heat and ACTH could cause physiological stress in the broilers. This physiological stress generated by the heat could modify other metabolic activities directly or indirectly linked to these two hormones.

Probably, the increase Ald in blood might allow maintaining a balance between water and minerals during heat stress. This could help broilers to retain Na⁺ in the body [17] and limit intestinal absorption of this mineral [26]. So in the heat, broilers were confronted with a dual stress, one of the higher temperature and the other to a fight against dehydration. During hot weather, the increase in Ald would be effective for the poultry for maintaining the balance of salt and water.

In conclusion, it seems that the hypothesis of the influence of intense heat on the main hormones was found. Indeed, chronic and continuous exposure of broilers to the HT had caused significant changes on the T3, T4, Ald and ACTH. Obviously, the element that triggered the hormonal and physiological changes was the chronic high heat. In these extreme thermal conditions broilers were able to adapt and resist through aldosterone and ACTH. Hormonal changes caused by HT persist until a late age of the animals. This indicates that broilers still needed Ald to face two problems: Resist the heat and drink enough water to dissipate this heat.

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